WHAT IS CLAIMED IS:

1. A method for increasing bone mass at least 10% in a host without a loss in bone strength or quality is provided that includes administering an effective amount of a compound that (i) binds to the estrogen α or ß receptor (or the equivalent receptor in the host animal) with an association constant of at least 10^8 M^{-1}, and preferably, at least 10^{10} M^{-1}: (ii) (a) induces estrogenic gene transcriptional activity at a level that is no greater than 10% that of 17ß-estradiol, and preferably no greater than 5, 1 or even 0.1% that of 17ß-estradiol when administered *in vivo* at concentrations of 10^{-11} to 10^{-7} M a dosage of at least 0.1 ng/kg body weight or *in vitro* in osteoblastic or osteocytic cells with natural estrogen receptors or cells transfected with estrogen receptors or (b) induces an increase in uterine weight of no more than 10% that of 17ß-estradiol (or the equivalent compound in a host animal); (iii) induces the phosphorylation of extracellular signal regulated kinase (ERK) when administered *in vivo* at a dosage of at least 0.1 ng/kg body weight or *in vitro* at concentrations of 10^{-11} to 10^{-7} M in osteoblastic cells with natural estrogen receptors or cells transfected with estrogen

receptors; and (iv) has an anti-apoptotic effect on osteoblasts at an *in vivo* dosage of at least 0.1 ng/kg body weight *in vitro* in osteoblastic or osteocytic cells with natural estrogen receptors or cells transfected with estrogen receptors.

2. The method of claim 1, wherein the compound is not an estrogen compound.

3. The method of claim 1, wherein the compound is an estrogen.

4. The method of claim 3, wherein the estrogen compound is converted to a nonestrogen by attaching a substituent which prevents the compound from entering the cell but does not significantly affect the binding of the compound to the estrogen cell-surface receptor.

5. A method for increasing bone mass at least 10% in a host without a loss in bone strength or quality is provided that includes administering an effective amount of a compound that (i)

binds to the androgen receptor (or the equivalent receptor in the host animal) with an association constant of at least 10^8 M^{-1}, and preferably, at least 10^{10} M^{-1}: (ii) (a) induces androgenic gene transcriptional activity at a level that is no greater than 10% that of testosterone, and preferably no greater than 5, 1 or even 0.1% that of testosterone when administered *in vivo* at a dosage of at least 0.1 ng/kg body weight or *in vitro* at concentrations of 10^{-11} to 10^{-7} M in osteoblastic cells with the natural androgen receptor or cells transfected with the androgen receptor or (b) induces an increase in muscle weight or virilization in women of no more than 10% that which is induced by testosterone (or the equivalent compound in a host animal); (iii) induces the phosphorylation of extracellular signal regulated kinase (ERK) when administered *in vivo* at a dosage of at least 0.1 ng/kg body weight or *in vitro* in osteoblastic cells with the natural androgen receptor or cells transfected with the androgen receptor; and (iv) has an anti-apoptotic effect on osteoblasts at an *in vivo* dosage of at least 0.1 ng/kg body weight or *in vitro* in osteoblastic cells with the natural androgen receptor or transfected with the androgen receptor.

6. The method of claim 5, wherein the compound is not an androgen.

7. The method of claim 5, wherein the compound is an androgen.

8. The method of claim 7, wherein the androgen is converted to a nonandrogen by attaching a substituent which prevents the compound from entering the cell but which does not significantly affect the ability of the compound to bind to the androgen cell-surface receptor.

9. The method of claim 1, wherein the compound also has a pro-apoptotic effect on osteoclasts at an *in vivo* dosage of at least 0.1 ng/kg body weight, or in osteoclastic cells with natural estrogen receptors or cells transfected with estrogen receptors.

10. The method of claim 7, wherein the compound also has a pro-apoptotic effect on osteoclasts at an *in vivo* dosage of at

least 0.1 ng/kg body weight, or in osteoclastic cells with natural estrogen receptors or cells transfected with estrogen receptors.

11. A method for selecting a compound that increases bone mass in a host at least 10% without a loss in bone strength or quality is provided that includes evaluating whether the compound (i) binds to the estrogen or androgen receptor (or the equivalent receptor in the host animal) with an association constant of at least 10^8 M^{-1}, and preferably, at least 10^{10} M^{-1}: (ii) (a) induces estrogenic or androgenic gene transcriptional activity at a level that is no greater than 10% that of 17β-estradiol or testosterone, and preferably no greater than 5, 1 or even 0.1% that of 17β-estradiol or testosterone, as appropriate, when administered *in vivo* at a dosage of at least 0.1 ng/kg body weight or *in vitro* in osteoblastic cells with the natural androgen or estrogen receptor or cells transfected with the androgen or estrogen receptor or (b) induces an increase in uterine weight of no more than 10% that which is induced by 17β-estradiol or muscle weight or virilization in women of no more than 10% that which is induced by testosterone (or the equivalent compound in a host animal); (iii) induces the phosphorylation of extracellular signal

regulated kinase (ERK) when administered *in vivo* at a dosage of at least 0.1 ng/kg body weight or *in vitro* in osteoblastic or osteocytic cells with the natural androgen or estrogen receptor or cells transfected with the androgen or estrogen receptor; and (iv) has an anti-apoptotic effect on osteoblasts at an *in vivo* dosage of at least 0.1 ng/kg body weight or *in vitro* in osteoblastic cells with the natural androgen or estrogen receptor or cells transfected with the androgen or estrogen receptor.

12. A method for screening for compounds that possess bone anabolic effects, comprising the steps of: a) contacting a sample of osteoblast cells with a compound; and b) comparing the number of osteoblast cells undergoing apoptosis in the compound-treated cells with the number of osteoblast cells undergoing apoptosis in an untreated sample of osteoblast cells.

13. A method for conferring bone protection on a population of cells in a subject through osteoblast/osteocyte anti-apoptotic effects, comprising the step of: administering an effective dose of a compound to said population of cells, wherein said

compound has a terminal phenol group and at least a second ring, wherein said compound has a molecular weight of less than 1000.

14. The method of claim ~~14~~ 13, wherein said compound has a molecular weight greater than 170.

15. The method of claim ~~14~~ 13, wherein said terminal phenyl ring is a non-steroidal compound.

16. The method of claim ~~16~~ 15, wherein said terminal phenyl ring is a phenolic A ring.

17. The method of claim ~~14~~ 13, wherein said effective dose of said compound results in a plasma concentration of less than 500 nM.

18. The method of claim ~~18~~ 17, wherein said plasma concentration is from about 0.02 nM to about 500 nM.

19. The method of claim 18, wherein said plasma concentration is from about 0.1 nM to about 1 nM.

20. The method of claim 13, wherein said compound is selected from the group consisting of a four-ring structure, a three-ring structure and a two-ring structure.

21. The method of claim 20, wherein when said compound is a four-ring structure, said effective dose is that which achieves a plasma concentration of less than 500 nM.

22. The method of claim 20, wherein when said compound is a three-ring structure, said three-ring structure is a phenanthrene compound.

23. The method of claim 22, wherein said phenanthrene compound is selected from the group consisting of a tetrahydrophenanthrene and an octahydrophenanthrene.

24. The method of claim 22, wherein said phenanthrene compound is selected from the group consisting of a phenanthrenemethanol and a phenanthrenecarboxyaldehyde.

25. The method of claim 20, wherein when said compound is a two-ring structure, said two-ring structure is fused.

26. The method of claim 25, wherein said fused two-ring structure is selected from the group consisting of naphthol and naphthalene.

27. The method of claim 20, wherein when said compound is a two-ring structure, said two-ring structure is non-fused.

28. The method of claim 27, wherein said non-fused two-ring structure comprises a linkage group.

29. The method of claim 13, wherein said compound is administered in combination with a reducing agent.

30. The method of claim 1, further comprising administering the compound in combination with a second pharmaceutical agent.

31. The method of claim 30, wherein the second pharmaceutical agent is bone anti-resorption agent.

32. The method of claim 30, wherein the second pharmaceutical agent is a bone mass anabolizing agent.

33. The method of claim 30 wherein the second pharmaceutical agent is an antioxidant.

34. The method of claim 30, wherein the second pharmaceutical agent is a dietary supplement.

35. The method of claim 30, wherein the second pharmaceutical agent increases the beneficial effect of the active compound on bone structure, strength, or mass.

36. The method of claim 30, wherein the second pharmaceutical agent is selected from the group consisting of an anabolic steroid, a bisphosphonate, a calcitonin, an estrogen or progestogen, an anti-estrogens such as raloxifene or tamoxifene, parathyroid hormone, fluoride, Vitamin D or a derivative thereof, or a calcium preparation.

37. The method of claim 30, wherein the second pharmaceutical agent is selected from the group consisting of alendronic acid, disodium clondronate, disodium etidronate, disodium medronate, disodium oxidronate, disodium pamidronate, neridronic acid, risedronic acid, teriparatide acetate, tiludronic acid, ipriflavone, potassium bicarbonate, progestogen, a thiazide, gallium nitrate, NSAIDS, plicamycin, aluminum hydroxide, calcium acetate, calcium carbonate, calcium, magnesium carbonate, and sucralfate.

38. The method of claim 5, further comprising administering the compound in combination with a second pharmaceutical agent.

39. The method of claim 38, wherein the second pharmaceutical agent is bone anti-resorption agent.

40. The method of claim 38, wherein the second pharmaceutical agent is a bone mass anabolizing agent.

41. The method of claim 38, wherein the second pharmaceutical agent is an antioxidant.

42. The method of claim 38, wherein the second pharmaceutical agent is a dietary supplement.

43. The method of claim 38, wherein the second pharmaceutical agent increases the beneficial effect of the active compound on bone structure, strength, or mass.

44. The method of claim 38, wherein the second pharmaceutical agent is selected from the group consisting of an anabolic steroid, a bisphosphonate, a calcitonin, an estrogen or

progestogen, an anti-estrogens such as raloxifene or tamoxifene, parathyroid hormone, fluoride, Vitamin D or a derivative thereof, or a calcium preparation.

45. The method of claim 38, wherein the second pharmaceutical agent is selected from the group consisting of alendronic acid, disodium clondronate, disodium etidronate, disodium medronate, disodium oxidronate, disodium pamidronate, neridronic acid, risedronic acid, teriparatide acetate, tiludronic acid, ipriflavone, potassium bicarbonate, progestogen, a thiazide, gallium nitrate, NSAIDS, plicamycin, aluminum hydroxide, calcium acetate, calcium carbonate, calcium, magnesium carbonate, and sucralfate.